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The ocean and especially the tropical ocean is, as it has always been, the great home of life. The forces which in cooler climates tend to repress and retard animal and plant life, here favor and force it onward. The battle for existence is here most incessant, its phenomena most marked. There results that boundless range of form and color, that exhaustless spring of individual life, which may well excite our wonder and our love.

THE ETIOLOGICAL CLASSIFICATION OF DISEASES.

BY FRANK S. BILLINGS.

BEFORE discussing this part of nosology it may be well to say a few words on the classification of diseases in general. First, we differentiate them according to that tissue of an organ in which the initial stage of the disease occurs, or in which the lesions predominate, or in other words, as to whether the active, or the stromatous, or supporting tissues are first complicated; that is, into interstitial or parenchymatous. It is well that this point be completely understood.

Those who are engaged in giving instruction in our medical schools, as well as those members of the profession who have an especial fondness for pathology, generally find that not only the students per se, but a great many practicing physicians, either have, or obtain, the idea that the above classification is something absolute, and that the processes are entirely limited to the tissues indicated in the differentiation. This is an error, but it seems to be a necessary failing of the text books to keep up this sharp differentiation, which would not be so serious had the authors but called attention to the fact that, while the pathological processes may begin in one kind of tissue, for instance the parenchymatous, if severe and prolonged in action they must eventually complicate the interstitial also. This condition of things is far more common in regard to the extension of interstitial disturbances to the parenchyma, than the contrary, especially if parenchymatous disturbances are of short duration. Again, it is far more common in some organs than others, For instance, a parenchymatous pneumonia may exist to an excessive degree without any proliferation to speak of occurring in the stroma of the lungs; or a chronic bronchitis, which is partially parenchymatous and partly interstitial as to the bronchial tubes, may be present without any very serious disturbance of the alveola-epithelium, but a well-diffused and active interstitial pneumonia cannot occur without the most severe parenchymatous complications resulting in the complicated parts, due principally to the encroachment of the interstitial processes upon the walls of the blood-vessels circulating through the interlobular spaces, thus leading to severe circulation disturbances, and, eventually, stasis in many parts of the lungs. The condition seen in contagious pleuro-pneumonia in cattle is a striking example of this kind of extension. On the contrary, it is very seldom that we have severe and prolonged disturbance of the active substance in the dense glandular organs, such as the liver or kidneys, without more less extension of the processes from the parenchymatous to the interstitial tissue, especially in the vulnerable spots around the larger blood-vessels, or where bifurcation of vessels is present. Hence it is better to look upon this classification as having reference to the tissues in which the disease began, or where the disturbances have been initially the most severe, rather than that they have been, or are, limited to a certain tissue, as the names selected appear to indicate.

Again, we classify diseases according to the product, the nomenclature being self-explanatory, such as, caseous, fibrinous, or catarrhal pneumonias; a fibrinous or a serous effusion; a hæmorrhagic, or purulent, process, and so on. Such other means of differentiation as acute or chronic, or the various critical sub-divisions, need not be here mentioned.

With these few and imperfect remarks as an introduction, let us turn our attention to the etiological classification.

On this point we have the altogether antiquated classification of diseases into "Contagious, infectious and sporadic," which should be replaced to-day by speaking of them as:

Extra-organismal, or exogenous;
Intra-organismal or endogenous; and,
Sporadic; that is of undemonstrable origin;

or, in other words, from the etiological point of view we can logically classify diseasesonly according to the primary origin of their specific cause.

A recent reviewer in the New York *Medical Record* says of my "Investigations of Cattle Diseases in Nebraska," which includes some researches made upon alcoholic material from yellow-fever patients, that said work "contains much that is interesting, but is, unfortunately, very controversial in tone."

What nonsense! How can one ever hope to establish truth in the face of error but by controversy? To my mind, the more grievous the error, the more grievous the necessity for correct controversy, and there are some very grave reasons in this country why the controversy should be turned into a very severe battle for the truth, so far as original research is concerned.

Going back to our original differentiation for a moment, Hueppe has come to the same idea when he speaks of the micro-organismal causes of disease as "obligatory parasites," by which he means that such etiological moments are primarily bound on the conditions offered by some form of animal life for their existence and continuous development; or, in other words, such diseases are "endogenous" in origin, to speak with Pettenkofer, or intra-organismal in origin, as I have termed it. An endogenous disease is one which, so far as we can historically trace its genesis, has found and still finds its locus of primary origin for each new outbreak or extension of the disease, in a diseased individual of some given species of animal life (and never in any other way); and then passes directly from the diseased individual to another susceptible healthy one, either by direct contact, or cohabitation, or by contact with some effluvia which has either come directly from, or been in immediate contact or relation with, such a diseased individual.

Speaking in the old sense, such a disease would be "contagious." Speaking acccording to the nonsensical usage of that word at present, no one can tell what its true origin might have been.

In contradiction to his obligatory parasitic diseases, Hueppe has given us the term of "faculative parasites," by which he means to indicate diseases of parasitic origin, but in which the focus of primary development (of the germs) is invariably outside the animal organism; still, that they have the faculty of living, for a time, within the organism of certain species of animal life, becoming parasitic or disease-producing for the time, when such animal organisms offer the necessary nutrient conditions to their life. To this class Pettenkofer has given the name of "exogenous," while I have termed them "extra-organismal," or diseases which found their primary origin in internal or surrounding conditions; or, in other words:

An exogenous disease is one which invariably finds its locus of primary origin not in, but outside of, an animal organism, that is, in the earth, or in the surroundings of animal life, where its micro-organismal cause develops under certain conditions of climate and soil which offer the necessary nutrients to its life and continuous development.

The infected earth or locality bears the same relation to animal life in the origin of exogenous disease that the infected animal organism does to healthy susceptible animals in endogenous diseases. That is, they each form centres of primary origin in their relation to specific diseases in their respective class, but with this difference: the focus of primary generation, or infection, is fixed in exogenous diseases, while it is movable in endogenous.

The *locus infectionis*, that is the point of primary infection or origin, is contagious in either case. In the one a healthy susceptible individual must come in direct contact, be upon or in such an infected locality, or come in contact with material derived directly from such a locality; while in an endogenous disease the same occurrences must take place in reference to some form of animal life. Hence it is to be readily seen that the word contagious has no logical use or place in the nosology of diseases, according to the results of modern investigation. Yet we find members of "Boards of Health" and other hygienic organizations, and physicians, rolling off the word "contagious" with the utmost nonchalance in connection with Actinomycosis, a disease due to a fungus which invariably finds its primary origin outside any form of animal life, and primarily infests some form of vegetation in our fields and pastures. We find such authorities speaking of this disease as "contagious," because, by intentional inoculation of the Actinomyces from diseased to healthy animals, it has been found that, under favorable nutrient conditions, it will continue its development in the same. This absurd idea, that contagiousness and transmissibility are one and the same thing, has found its chief support in the teachings of Robert Koch and his school. So long as Koch keeps himself confined to the exact observational and descriptive ground of bacteriology his reputation is unquestionable, but the moment he touches the field of general pathology he shows the utmost incapacity for his work, and, as intimated above, has done more to mislead the medical profession of to-day regarding a logical understanding of the true nature of diseases genetically, than all other writers combined. Even the great authority of the world's greatest epidemologist, Pettenkofer, and the generally sharp and logical teachings of Hueppe, seem to have been utterly unable to stem the tide of misconception set flowing in full flood through the teachings and example of Robert Koch and his school.

They have spoken of the "contagium" of anthrax, black-leg, cholera, schweineseuche, and such diseases, all of which are faculatively parasitic; all of which find their locus of primary origin invariably outside of any and every animal organism.

Why?

Because such diseases are transmissible to healthy from diseased animals by artificial (or accidental) inoculation. The same is true of endogenous diseases if we have susceptible animals.

Anthrax can be transmitted to a man from the dirt where a diseased sheep, or ox, may have been buried. Syphilis can be transmitted from a diseased to a healthy man by an accident with a scalpel. What is the difference, then? According to Koch and his followers they are both alike. They are, in one sense, both infectious diseases, but they are vastly different in point of origin, the one originating in and from infected earth, the other in and from a diseased individual. The source of primary origin alone decides this question. Accidental or experimental extension has nothing whatever to do with it.

But take another case which at the outset looks somewhat complicated. An ox is confined in a stable; it becomes diseased with anthrax; a fly stings it and then lights on the attendant and bites him; the man dies of malignant pustule. In this case the diseased ox was "contagious" to the fly and the latter to the man, but is anthrax a contagious disease? By no means; the fly was the accidental transmitter, the living syringe which filled itself from the blood of the ox and then introduced the infection to the man by its needle-like proboscis. The ox, however, became primarily infected from some source having no connection with animal life.

These statements of the case as to "contagion" and "contagiousness" should settle the question in every logical mind.

Remember, transmissibility of an inficiens either artificially or accidentally has nothing to do with the differention of infectious diseases as to class. The locus of primary origin and continuous natural development can alone decide as to whether a given disease is exogenous or endogenous in character.

Let us return to the question of the pathogenic nature of actinomycosis again for a moment. Actinomycosis is not an infectious disease in any true sense of the word, every authority to the contrary notwithstanding. An infectious disease, be it exogenous or endogenous in character, is one which is invariably accompanied at some time in its course by one general symptom, —fever, which is an invariable symptom of general or constitutional disturbance. The very word infection means that something has been produced which is of a general polluting character to the infected organism. Nothing of this kind occurs in actinomycosis per se. There is not necessarily any fever in this disease. Actinomycosis is always local, never general. Unless the disturbances are of such a character, or so situated, as to mechanically interfere with the functions of the organism, the individual does not necessarily emaciate. Aside from pulmonary actinomycosis the invasion is dependent on the presence of some lesion in the mouth or pharynx, be it a diseased tooth or distended ostia to glands or ducts. Not even a diseased tooth is alone sufficient. There must be follicular disease also, or the actinomyces do not gain entrance. Wherever the symptoms of general infection are present in man or animal,—far more common in man,—it will be invariably found that the invading actinomyces are accompanied by one or more of the pus-producing cocci, *Streptococcus pyogenus albus* or *aureus*, or some really infection-producing organism. In such a case, then, we have a "mixed invasio-infectious pathological complex." I do not believe that a single case of actinomycosis in man was ever necessarily due to the presence of a diseased animal. Men have been exposed to a common cause, that is all, and have unfortunately presented some lesion which acted as an atrium to the actinomyces. A recently reported case where a veterinarian is said to have been dressing a "big jawed steer," and then scratched his own tongue with his thumb nail and acquired actinomycosis linguæ (if true), was but a fitting punishment for so filthy an individual, and proves nothing but the possibility of accidental transmission, which was known well enough previously without such a disgusting and crucial test.

If actinomycosis is so "contagious," what about the thousands of horses pastured annually and stabled with "lumpy jawed" cattle? If so terribly dangerous to man, why do we find so few cases when hundreds of men, not one with sound mouths, have been in contact with diseased cattle for years?

Another class of diseases of exogenous origin, but which differs from that previously considered, is known as malarial infectious diseases. They differ from all other exogenous diseases in that, while, like the others, local in origin, they always remain local; their cause cannot be transported in the body of a diseased individual, and secondary centres of infection be caused thereby, nor have they ever been transmitted by inoculation. Febris intermittens is a striking example of this class.

As of other exogenous diseases the micro-organismal cause of malarial infectious diseases also belongs to Hueppe's class of "faculative parasites," yet one is surprised and almost shocked to see that an observer generally so logical as Hueppe, and one for whom I have the greatest admiration and respect, should, in a late article upon the "Etiologie der Cholera Asiatica" (*Berliner Klinische Wochenschrift*, No. 9, 1890) speak of this disease, of which he describes the cause to be a "faculative parasite," in the following absurd, illogical, and most decidedly contradictory manner, when he says, "*Die Cholera asiatica ist wirklich eine miasmatisch-contagious Krankheit*,"—Cholera Asiatica is truly a miasmatic-contagious disease. A "miasmatic-contagion" is an unsupposable thing. A disease of faculative-parasitic origin cannot be contagious in any logical sense. The parasite finds its primary focus of development invariably outside of any animal organism, or it could not be faculative; and hence cannot be contagious. When will this absurdity be driven out of the minds of otherwise close observers and logical thinkers? A person affected with intermittent fever is neither dangerous to others, nor can such an individual infect new localities,—at least neither of these phenomena has ever been observed in the history of such diseases.

INVASION OR MIXED INFECTION.

The time has certainly come in connection with micro-organismal-etiological research where most exact attention must be given to the pathological differentiation of germs which are not in any direct way connected specifically with the etiology of a given disease, and yet may be the cause of secondary or complicating lesions. Although I have not as yet published anything in detail upon this subject, still it has called for a large amount of very exact study and much experimentation. The one question which has been entirely overlooked is, that if an investigator desires to decide upon the specific infectious quality of a given germ when he finds it as a complicating phenomenon in a given disease caused by a well-known micro-organism, he must make his tests in that species of animals in which he found it, and not in any others. It must be understood that by infection we can only mean one thing, and that is the pollution of the blood by some specific septic or toxic producer, let it be that that specific producer finds general distribution over the organism and develops or carries on its work in the blood, let it be that the producing organism is locally confined to its point of introduction (as in rabies, tetanus, etc.), where it produces the toxic (or septic) material, and from whence it is taken up by the circulation (lymphatic or blood) and distributed over the system, or let it be produced in some organ from whence the same effect is produced in the same way as in Asiatic cholera. Infection means the pollution of the system by something produced either generally, or locally, within it, and presupposes the continued production of such material for a given period by the producer, and its accumulation in the system both as a septic material and chemical irritant.

I cannot accept a late attempt at differentiating diseases in which the production of such an irritant takes place from the point of entrance to the organism as intoxicating diseases (tetanus, rabies), in contradistinction to septicæmic diseases, where the germs do not remain local. It might be well to call these forms of infection toxæmic, in order to distinguish them from the strictly septicæmic, where the polluting micro-organisms develop in the blood itself and thus pollute it. We have also others in which both phenomena take place, of which anthrax is an example. For me, intoxication diseases are those in which the organism is saturated by a given poison, which may be introduced in any way, but which is not generated either by any part of itself, or any parasitic organism which has been introduced. Poisoning, in the common acceptation of the term, is intoxication, let it be by morphine, nicotine, alcohol, or what not. Intoxication does not presuppose a poison-producer lodged in the organism. Infection does. Naturally, such a definition has no reference to uræmic or physiological intoxication, the nature of which should be self-evident. This form can well be called intra-organismal or cumulative intoxication, while the first-named could be termed extra-organismal, keeping in view our previously-considered classification of infectious diseases. Or, to speak with Hueppe, the one is obligatory intoxication (when the kidneys do not act), the other faculative, or acquired by the individual. Or, in other words, the one form cannot be presented by the individual, hence is obligatory, while in the other he acquires the "faculty" of intoxicating himself.

To return to invasive complications: Correctly speaking, every lesion of parasitic origin must be considered as invasive in which specific pollution of the blood is not directly produced by the invader.

It has been said, and must be repeated, that the experimental test of this point must be made on healthy individuals of the same species in which the parasite was discovered, and if a micro-organism, by sub-cutaneous injection only.

I insist on the latter point with as much intensity as on the others that have been raised.

For instance, there are many micro-organisms found in the respiratory tract of animal life, or in the intestines, some of which under circumstances cause local lesions, and yet not one of them need necessarily be specifically infectious in or to the species of animal in which it was discovered. On the other hand, they may be violently so—septicæmic in rabbits, mice, guinea-pigs, or some of the many animals used in experimentation, the celebrated sputum coccus for example, and the majority of such organisms lately described by Miller in his work on "Bacteria in the Mouth." There are quite a number of such in the intestinal canal of hogs, but of those which I have thus far tested on rabbits, guinea-pigs, mice and ground-squirrels, and found fatal to one or the other, not one affected pigs in the least in very large amounts when introduced in the same way; that is, sub-cutaneously.

This class of germs is not specific in any sense of the word, according to our present knowledge. Not one of them is known to be the cause of a natural infectious disease in any of the animals in which they have been shown to have septic action by experimental inoculation. They should not even be called "pathogenic," as has been the case, as the word is misleading, and has too much of a specific sense to be used in this connection.

To any mind the only logical term to apply is, experimentally-infecting (not disease, but) micro-organism.

It may be asked why I have not reported upon my experiences with this class of germs? To which I reply, that once I had no interest in them, and not until circumstances made it necessary to bother with them did I give them any attention. Just at present they are having a "cumulative action," to be exploded with an intoxicating effect in certain directions when a suitable time comes.

It is my opinion that the U. S. Government "Swine Plague" is one of those cases of "mixed invasion," and I base that assertion on the fact that those who have tried have utterly failed (according to their published evidence) in sickening or killing a single hog by subcutaneous injection. I utterly ignore results following the injection of 8 or 9 ccm. of a bouillon culture directly into the lungs of a healthy animal. Just how many varieties of germs (absolutely non-specific) would cause pneumonia, and even gain entrance to the circulation and find more or less general distribution over the organism thus maltreated, can be determined by embryo experimenters; but it can be answered beforehand, Just as many as find suitable conditions for continued development in the conditions thus offered.

Welch himself says that not a single epizootic of his Swine Plague has been seen in this country. Then why has he insulted common sense and pathological knowledge by speaking of such an insignificant and completely undemonstrated complication as a new "Swine-Plague?"

It would be equally justifiable to speak of a large bacillus I shall speak of at another time as the cause of certain embolic hæmorrhagic lesions in the kidneys, seen sometimes in Swine-Plague, or as a specific or mixed infectious nephritis, because it often causes a mechanical-foreign-body-pneumonia for the same reason. It is not a specific germ. It does not cause infection. It is an invasion. The germ itself is absolutely harmless from the infection standpoint. It would be as sensible to speak of the filaria broncho-pneumonias in cattle, sheep, or hogs, as infectious, as those caused by these non-polluting germs.

There are many pulmonary complications of germ-parasitic origin of this invasio-non-infectious type, which occur in the lungs, which have not received the attention their merits deserve. In diseases of a specific acute infectious character (exogenous in origin especially), such as typhus abdominalis, the true Swine-Plague, the corn-stalk disease, one of the chief phenomena is a very high fever, and the most essential lesions of such diseases are the extreme parenchymatous changes in the muscles, and dense glandular organs such as the liver and kidneys. Added to these, and of equal importance, are similar changes in the heart-muscles, and the disturbance of the secretive functions of the kidneys. Briefly speaking, the elements of these organs are swollen, their condition, as a whole, is more or less anæmic, due to the pressure of the swollen cells upon the delicate capillary ramifications. These pathological disturbances necessarily lead to pressure, *à vis à frontis*, upon the circulation towards the points of least resistance; that is, to an accumulation of the blood in those organs the structure of which offers the best support to the blood-vessels, and thus favors their distention and engorgement. The two points are, first, the lungs, and next in importance the intestines. The lungs, however, chiefly deserve our attention. This condition of engorgement finds additional support in the weakened propelling power of the heart, due to the parenchymatous disturbance of the muscles. Again, the prostrate position of the individual [in man] in such cases, the inability, or non-desire for movement in both man and animals, increases this condition in the lungs, because the muscles are not called upon to take up any nutrition, and hence all conditions favoring diffusion of the blood are partially stagnated. In such cases what may be termed stagnation-pneumonia (or hypostatic pneumonia—a less correct term) is the physiological result; some might prefer to term it pathological. We will not discuss straws. In such a case we have another very vascular tissue to consider, and that is the mucosa of the bronchioles; this also is engorged, and as in the lung a serous effusion takes place into the alveoli, so in the bronchioles, the same occurs, leading to a complete obstruction of the circulation of the air;

hence atalectosis, then pneumonia; but, in this case, assuming that no micro-organisms are present in the tubes, the cellular plugging up of the alveoli does not begin in those adjoining the tubes, but in those on the extreme limits of the infundibulus. The wonderful provisions of nature are nowhere more marvelously displayed than in the protective function of the cilia of the bronchial mucosa; so long as the mucosa remains comparatively normal, these delicate hairs move all foreign substances, including a surplus of secretion, towards the larynx, whence they are expectorated. This has reference to all forms of micro-organismal life as well. But when the mucosa becomes severely engorged, as in the case in point, the movements of the cilia become heavy and retarded, and when the engorgement of the vessels is so severe as to lead to serous effusion, these movements become completely paralyzed, the natural result being the retention of any foreign elements present at the point where they are caught. This serum and the existing temperature offer the most favorable conditions for the development of any germs which may be present. It can be truly said that in such a case we have a sort of natural "Esmarli Suhe." If such germs are caught at the termination or near there, or along the course of the most delicate bronchioles, they at once commence to develop into colonies, and soon fill up the tube, causing an irritation of the embracing alveolal walls, and set the epithelium of the same into active proliferation, the natural result being broncho-pneumonia. This pneumonia will depend for its character on the specific nature of the germs, which may be simply mechanically irritative in their action, purulent, or gangrenous. According to my experience, the first is the most common variety. In such a case, then, the individual has a pretty poor chance for life. To an already begun stagnation pneumonia is added one of a bronchial type, or vice versa. Again, in such an invasive broncho-pneumonia, the germs soon find their way into the circulation, either by the lymphatics or through penetrating the capillaries, most probably the former, and in such cases we naturally would find a mixture of micro-organisms in the blood.

As said, this occurs in typhus abdominalis. How many times has a physician congratulated himself and his patient that the "typhoid" was over, only to come the succeeding morning to find the temperature again elevated, the face cyanotic, and the patient breathing rapidly and with difficulty. Surely the diagnosis was correct the previous day, and the prognosis justified? What then has happened?

1st. Stagnation pneumonia.

2d. Friedländer's bacillus or Fränkel's pneumococcus, or some other micro-organism, has been caught in the bronchial tubes, as suggested, and the unfortunate patient is again the object of bacterial attack.

As said so many times, the real pathogenic question is, Are the organisms (or germs) at the bottom of this deuteropathic complilication simply invasive in character, or has a secondary infection taken place? It is of the utmost prognostic importance that investigators truly inform practitioners which of these two is most likely to be the case.

Though not a a clinician, it would seem as if more attention ought to be given to the movements of the limbs by attendants, or massage treatment in such cases by physicians. Of the value of forced movement in an exactly similar case in Swine-Plague, I can speak most emphatically. It will save an otherwise fatal cases, and not only one case, but will lessen the percentage of mortality in a herd. I have proved this by actual experimentation in two different bunches of hogs inoculated at the same time with the same dose of virus. The ones forced to move several times a day, and as actively as they could, with a whip, all lived. The others, kept closely confined all the time from the day they were inoculated, all died.

It has been said that the larger number of these deuteropathic pulmonary lesions of micro-organismal origin will be found to be simply invasive, and due to mechanical irritation. In Swine-Plague, I know of three distinct micro-organisms which thus cause an invasive broncho-pheumonia, only one of which has any experimental virulence in small animals, and neither of them in swine, on subcutaneous inoculation. In the "Corn-Stalk Disease of Cattle," an unquestionable intestinal infection, several varieties have been found which caused broncho-pneumonia, and, so far as

I have pursued them, have no experimental virulence whatever in any of the usual small animals. The large bacillus (already spoken of) which causes embolic hemorrhage in the kidney in some cases of Swine-Plague, also causes broncho-pneumonia in diseased swine, but is harmless otherwise. The specific disease supplies the field, or opens the way, for the action of these adventitious germs through the disturbances of the circulation. That similar lesions may be due to microörganisms which also find their way into the circulation from the intestinal tract I have no doubt, though I am utterly without experience in that direction.

As a termination to this part of my story I will again repeat: It is absolutely necessary to determine whether these deutero-micro-organisms are simply invasively-mechanical in their action, or which of them also cause secondary infection.

THE SILVER LAKE OF OREGON AND ITS REGION.

BY E. D. COPE.

IT took me three days and a quarter to ride to Silver Lake from Fort Klamath, by an indirect road. We came by Sprague river and Siacan Valley. The former runs into Williamson's River and into Lake Klamath.

Before I left Fort Klamath, the soldiers brought in a splendid lot of trout from these rivers, and I secured several in alcohol. They were the *Salmo purpuratus* Pallas, and I have described their numerous variations in the Philadelphia Academy Proceedings for 1882. The largest weighed twelve pounds.

Next day I started out with a four mule team and wagon with provisions. I rode a cavalry horse, "Jim," a heavily-built gray, with a good outfit of saddle-bags and straps. They gave me an old Klamath chief as a guide, from the Agency near by. This was